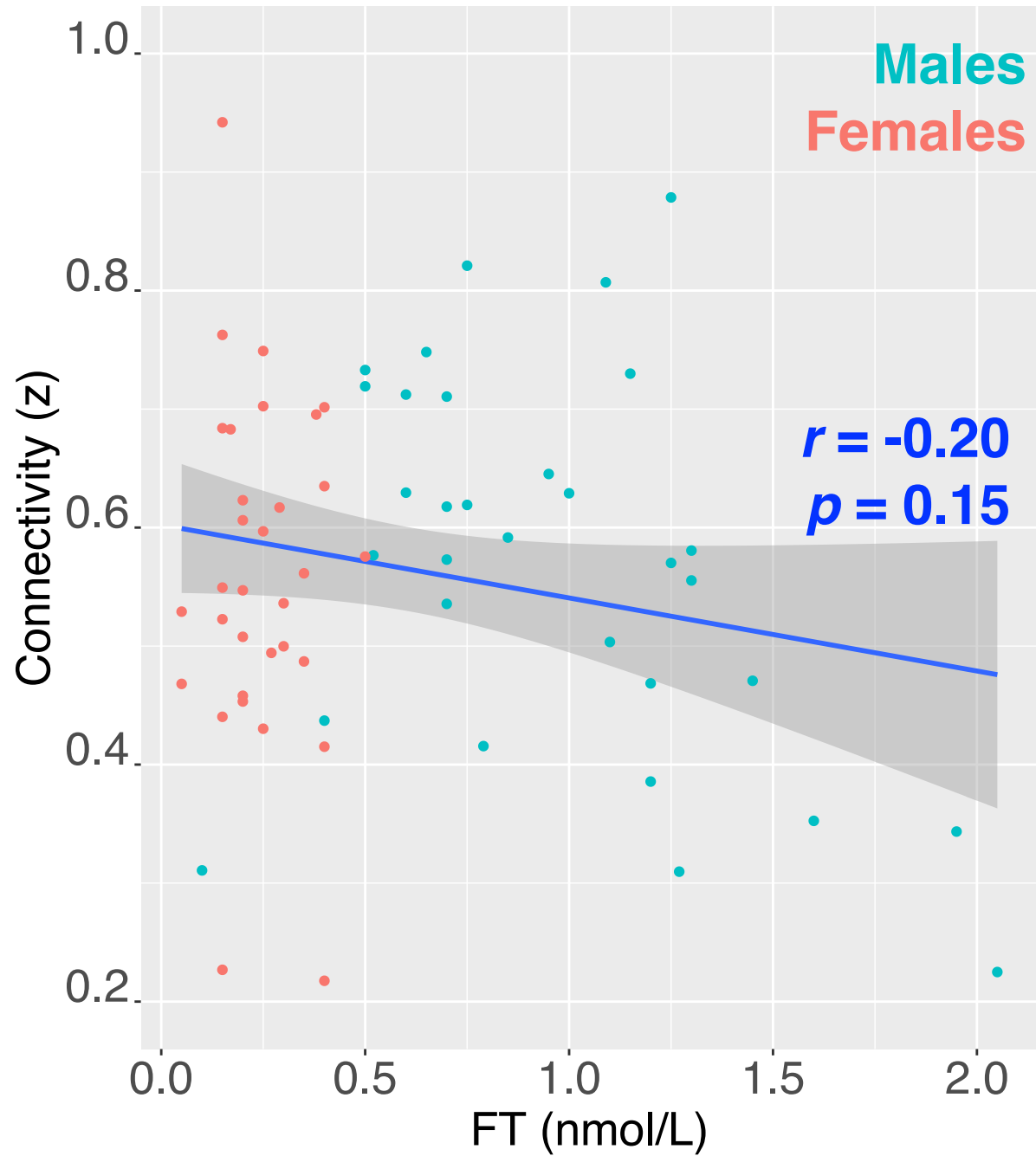
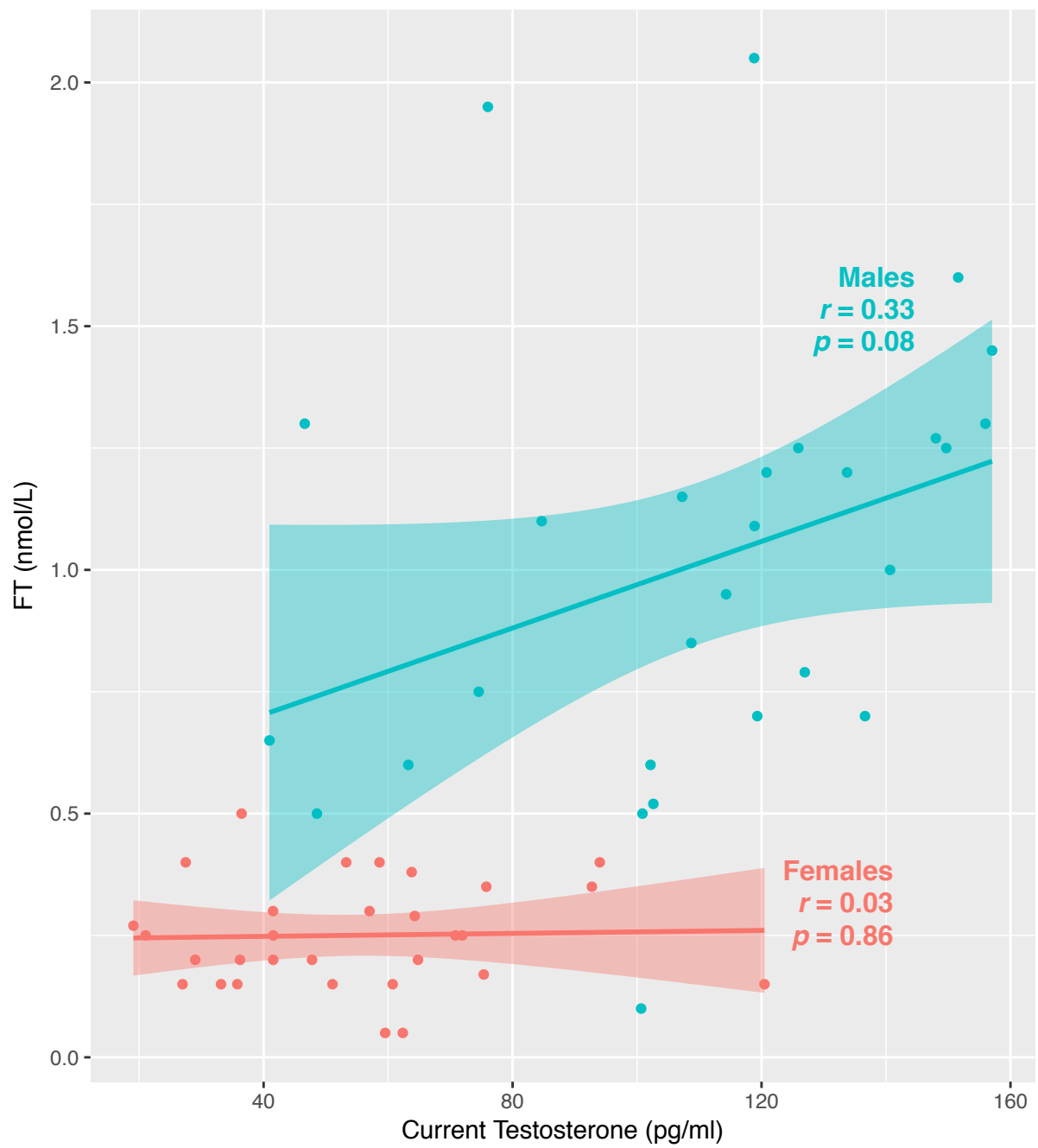


*Supplementary Results and Figures*

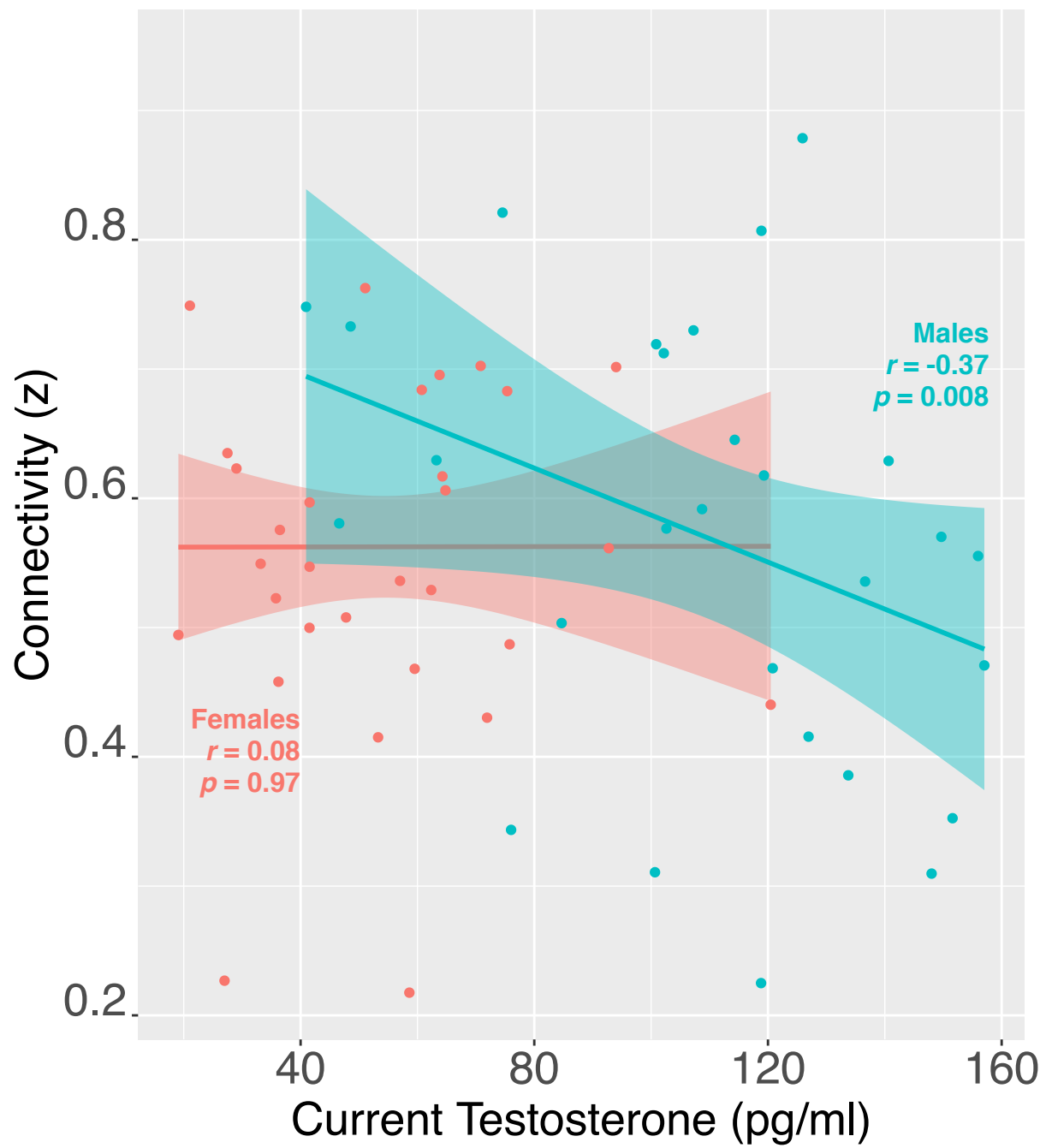
*Supplementary Figure 1: Association between FT and DMN connectivity ignoring sex.*



**Supplementary Figure 2:** Relationship between FT and current testosterone level in adolescence.

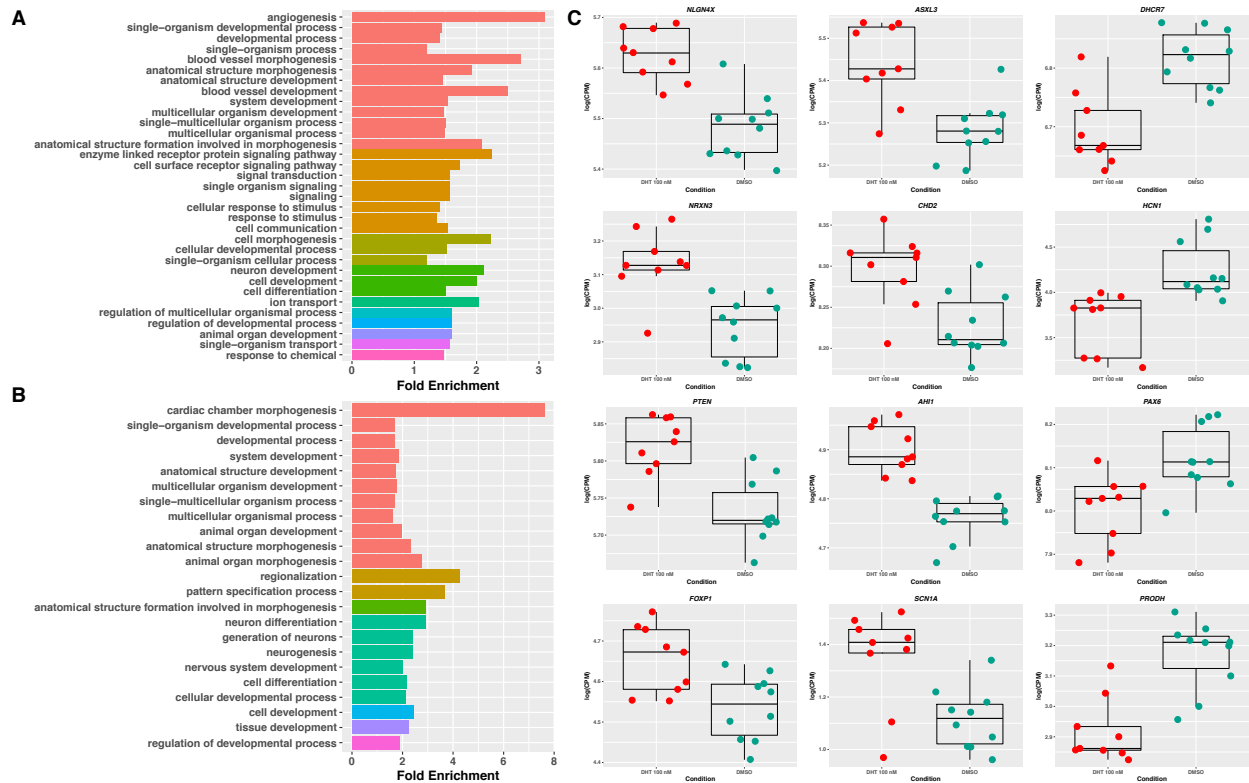


*Supplementary Figure 3: Relationship between current testosterone and DMN connectivity*

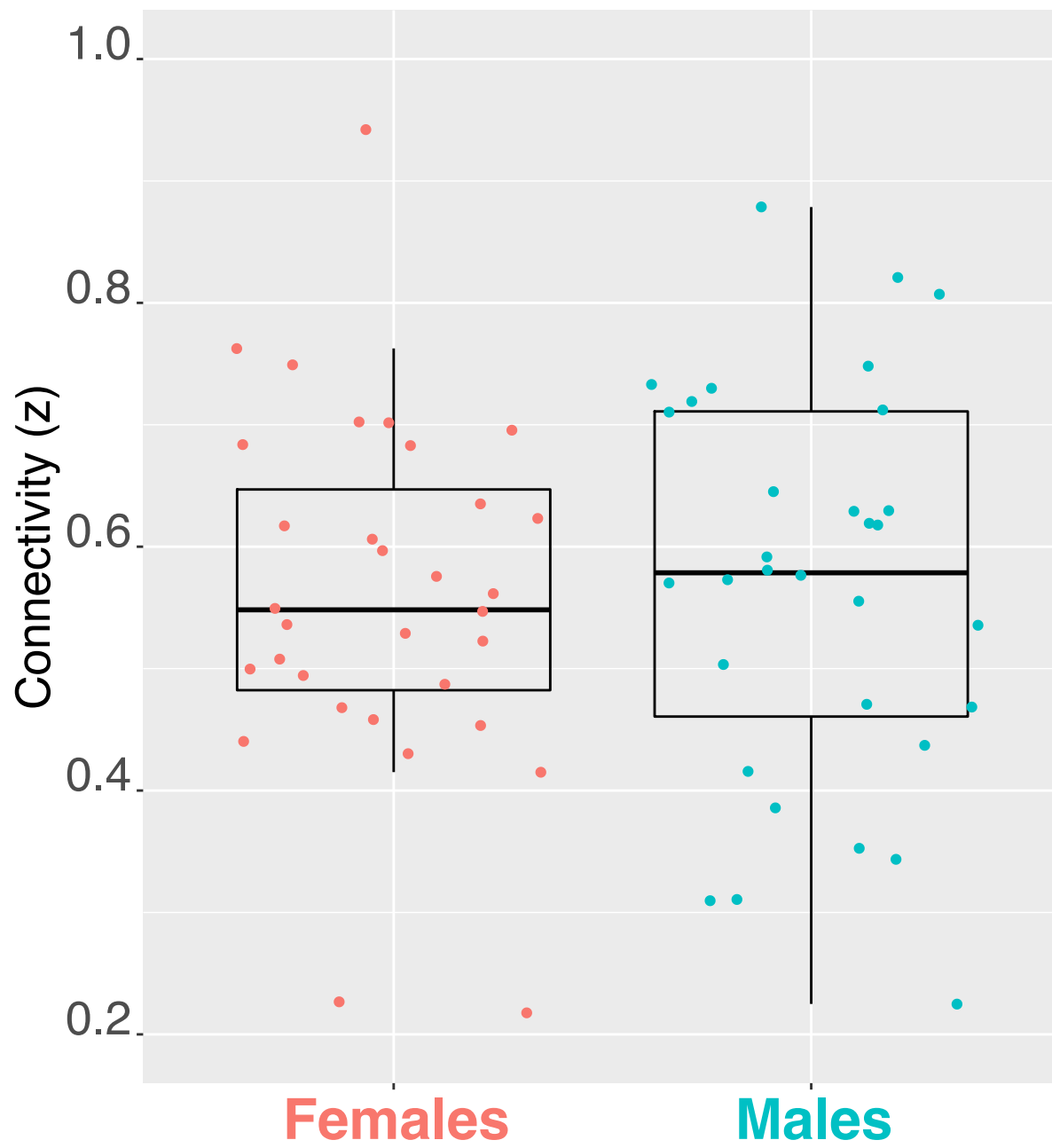


**Supplementary Figure 4: Biological process enrichment of DHT-upregulated (A) and downregulated (B) genes sets and plots of specific autism-associated genes (C).**

Quartier and colleagues recently reported DHT dysregulates genes were enriched for autism-associated genes<sup>1</sup>. The DE analysis in this paper utilizes the same RNA-seq dataset, but performs the DE analysis differently than that of Quartier and colleagues. Therefore, we re-examined this question with the current study's DE lists. Upon examining SFARI Gene Scoring categories for overlap with autism-associated genes, we find that a number of genes in the 'Syndromic' category are enriched in genes that are influenced by DHT manipulation (OR = 2.16,  $p = 0.02$ ). These genes include *AH11*, *ASXL3*, *CHD2*, *DHCR7*, *HCN1*, *MEF2C*, *PAX6*, *PRODH*, *PTEN*, and *SCN1A*. *ASXL3* and *PTEN* are also classified in the high-confidence category. No other gene scoring categories were enriched. However, there were many other notable autism-associated genes influenced by DHT, including *NLGN4X*, *NRXN3*, *FOXP1*, and *SCN9A* (Supplementary Figure 4C). Thus, this analysis highlights a largely-overlapping finding with the prior paper by Quartier et al.,<sup>1</sup> showing that many syndromic causes of autism are enriched within the DHT-dysregulated gene set.



*Supplementary Figure 5: DMN connectivity across males and females*



## References

1. Quartier A, Chatrousse L, Redin C, Keime C, Haumesser N, Maglott-Roth A *et al.* Genes and pathways regulated by androgens in human neural cells, potential candidates for the male excess in autism spectrum disorders. *Biol Psychiatry* 2018.